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# Chronic Effects of Cadmium on Two Species of Mysid Shrimp 'Mysidopsis bahia' and 'Mysidopsis bigelowi'

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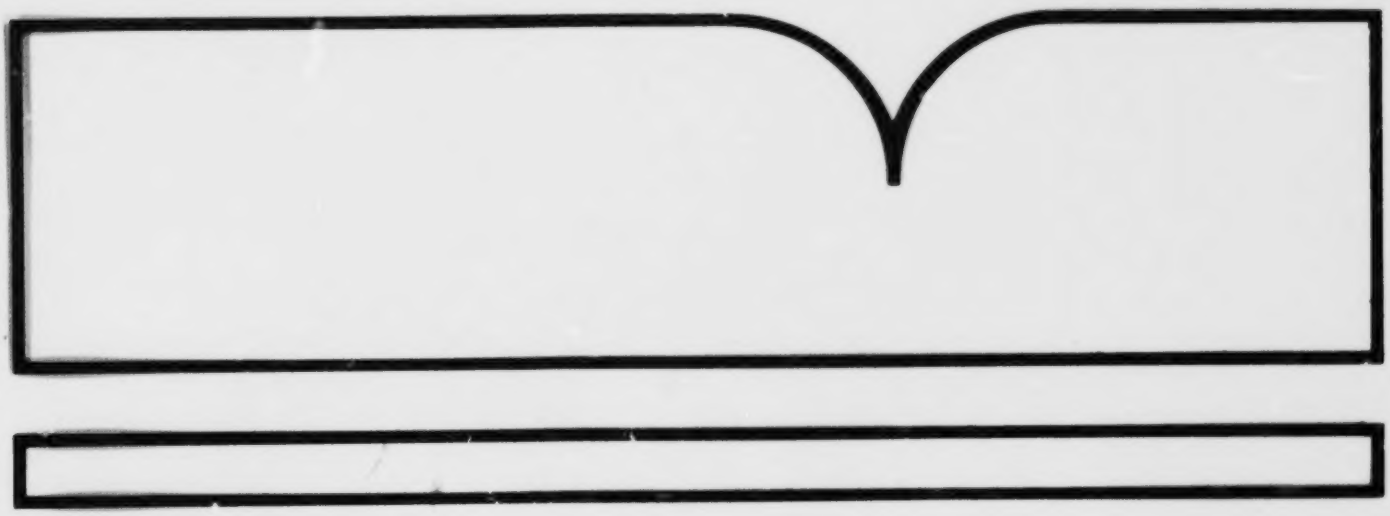

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16. ABSTRACT

Two species of mysid shrimp, the sub-tropical *Mysidopsis bahia* and the northern temperate *Mysidopsis bigelowi*, were exposed simultaneously to cadmium (as $CdCl_2$) in a continuous-flow bioassay system to determine the effect on survival and reproductive success. Temperature and salinity were maintained at 21 ± 1°C and 30‰, respectively. The 96-h LC50 was 110 $\mu g\ \ell^{-1}$ for both species. The 23-day life cycle LC50 for *M. bahia* was 19.5 $\mu g\ \ell^{-1}$ and for *M. bigelowi* the 27-day LC-50 was 14.8 $\mu g\ \ell^{-1}$. At 10 $\mu g\ \ell^{-1}$ a series of morphological aberrations were observed in both species at the onset of sexual maturity. Carapace malformations apparently prevented molting after the release of the initial brood and resulted in death of brooding females. As a result, although the initial reproductive rate at this concentration was successful, successive broods could not be produced. For both species in this study the no observed effect concentration was 5.1 $\mu g\ \ell^{-1}$; the effect concentration was 10.0 $\mu g\ \ell^{-1}$. Mechanisms were postulated in this study to explain the effect of cadmium on the molting process and on calcification and enzymatic reactions of osmosis.

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# Chronic effects of cadmium on two species of mysid shrimp: *Mysidopsis bahia* and *Mysidopsis bigelowi**

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Keywords: Mysidacea, cadmium, toxicology, opossum shrimp, *Mysidopsis bahia, Mysidopsis bigelowi*

**Abstract**

Two species of mysid shrimp, the sub-tropical *Mysidopsis bahia* and the northern temperate *Mysidopsis bigelowi,* were exposed simultaneously to cadmium (as $CdCl_2$) in a continuous-flow bioassay system to determine the effect on survival and reproductive success. Temperature and salinity were maintained at 21 ± 1°C and 30‰, *respectively. The 96-h LC50 was 110* $\mu g\ l^{-1}$ for both species. The 23-day life cycle LC50 for *M. bahia* was 19.5 $\mu g\ l^{-1}$ and for *M. bigelowi* the 27-day LC-50 was 14.8 $\mu g\ l^{-1}$. At 10 $\mu g\ l^{-1}$ a series of morphological aberrations were observed in both species at the onset of sexual maturity. Carapace malformations apparently prevented molting after the release of the initial brood and resulted in death of brooding females. As a result, although the initial reproductive rate at this concentration was successful, successive broods could not be produced. For both species in this study the no observed effect concentration was 5.1 $\mu g\ l^{-1}$; the effect concentration was 10.0 $\mu g\ l^{-1}$. Mechanisms were postulated in this study to explain the effect of cadmium on the molting process and on calcification and enzymatic reactions of osmosis.

## Introduction

*Mysidopsis bahia* is found in sub-tropical coastal waters, principally in the Gulf of Mexico (Molenock 1969; Odum & Heald 1972). *Mysidopsis bigelowi* is a northern temperate species with a geographic range along the entire eastern United States coast and in the shallow shelf waters of Georges Banks, the valuable fishing area off New England. Ecologically, both species are major food sources for bottom feeding fishes such as striped bass *Morone saxatilis* (Markle & Grant 1970), silverside *Menidia beryllina* (Odum & Heald 1972), and several species of flounder (Stickney *et al.* 1974).

The acute toxicity of cadmium and its compounds has been determined for a wide variety of marine species (US EPA 1980). The sublethal effects of cadmium have been less extensively studied. These studies are often restricted to a particular life stage of a limited number of test species (Vernberg *et al.* 1977). Many sublethal studies involve assessment of the synergistic effects of cadmium and environmental factors such as salinity and temperature (O'Hara 1973; Jones 1975; Rosenberg & Costlow 1976; Theede *et al.* 1979). Chronic exposure of marine animal species to cadmium through an entire life cycle are limited to studies with two invertebrates. D'Agostino & Finney (1974) found that with the harpacticoid copepod, *Tigriopus japonicus,* the development of ovigerous females was inhibited by chronic exposure to cadmium. Nimmo *et al.* (1977) exposed *M. bahia* throughout its life cycle and reported delays in brood formation and a decrease in the number of young under varying ambient temperature and salinity regimes.

The objectives of this study are to evaluate the suitability of *M. bigelowi* for use in chronic, life

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cycle toxicological studies and to compare the chronic toxicity of the congeneric species, *M. bahia* and *M. bigelowi*. As a further comparison, we contrasted our study with *M. bahia* (conducted at 21 °C and 30 ‰ salinity) with a similar study (Nimmo *et al.* 1977) at 20–28 °C and 15–23 ‰ salinity. This comparison permits the evaluation of the significance of naturally occurring environmental variables on both the acute and chronic responses of *M. bahia*.

## Methods and materials

Stock cultures of *Mysidopsis bahia* were supplied by the US EPA Environmental Research Laboratory at Gulf Breeze, Florida. *Mysidopsis bigelowi* were collected from Ninigret Pond, Rhode Island, in May 1980.

Animals were cultured in flow-through 76 ℓ glass aquaria with undergravel filters and a dolomite substrate with 2.5 cm of beach sand prewashed in deionized water and muffled at 500 °F for 6 h. Cultures were continuously supplied with filtered (15 μm) natural seawater (21 ± 1 °C and 30 ‰ salinity) at a rate of 100 mℓ/min, resulting in 99% volume exchange every 24 h. The outflows were covered by Nitex* screen (250 μm) to prevent the escape of mysids or food organisms.

Forty-eight hour old nauplii of *Artemia salina* were added as food organisms at the rate of $7 \times 10^4$ nauplii/day for each 76 ℓ culture.

Continuous-flow bioassays were conducted in an exposure system described in Sosnowski *et al.* (1979). The exposure system employed a siphon-flush mechanism that produced a 50% exchange of volume every 4 h. Incoming flow rate was approximately 30 mℓ/min; outflow was 100 mℓ/min every 4 h. Bioassays were maintained at a temperature of 21 ± 1 °C, 30 ‰ salinity, and illumination of 1000 lx on a 12L:12D cycle.

Acute and life cycle toxicity tests were initiated with 24 h old juveniles which were randomly distributed into six replicates of five animals each in four toxicant concentrations and a control. Each replicate was fed 48 h old *A. salina* ($7 \times 10^2$ day$^{-1}$). Acute 96-h toxicity assays were conducted according to ASTM Standard Methods (ASTM 1980). In the life cycle test, males and females at sexual maturity were redistributed among replicates at each concentration to provide an equal male: female distribution.

The following reproductive parameters were determined to be the best indicators of pollutant stress: sexual maturation time, first appearance of eggs in the brood sacs, first brood release, the number of broods released per female, and average number of young per brood.

To normalize differences in the number of ovigerous females available per exposure concentration, the number of available female reproductive days (AFRD) was calculated by multiplying the number of sexually mature females by the number of days each survived. Two estimates of productivity were calculated using the following ratios: number of young produced per available female reproductive day and number of broods released per available female reproductive day. Analysis of variance procedures were applied to these ratios to determine if there were statistically ($P = 0.05$) significant differences between treatments. Differences were identified using Dunnett's test (equal variances) or Behren's-Fisher test (unequal variances) (Snedecor & Cochran 1980). The Kruskal-Wallis Test (Lee 1980) was used to test for differences between species.

Cadmium (primary standard for atomic absorption; $CdCl_2$) was used as the toxicant in all assays. Toxicant stock solutions were prepared in deionized water at one hundred times the desired final concentrations. The stock solutions were metered at a constant rate and proportionally diluted (1:100) with 15 μm filtered seawater. The flow rate of dilution water was controlled with capillary tubing to produce nominal cadmium concentrations of 0.0, 5.5, 25.5 and 55.0 μg ℓ$^{-1}$. Water samples from the control and each dosed concentration were analyzed twice weekly by heated graphite atomization atomic absorption (HGA-AA). All cadmium determinations were made with a Perkin-Elmer Model 360* atomic absorption instrument coupled to a Perkin-Elmer Model 2100* heated graphite atomization unit (Gerald Hoffman, personal communication). The reproducibility of each analysis varied from 100% at the detection limit to 10% at the 5 μg ℓ$^{-1}$ concentration to 5% at the 50 μg ℓ$^{-1}$ concentration.

*Mention of commercial products does not constitute endorsement by the Environmental Protection Agency.

Measured exposure concentrations were. ≤0.3, 5.1 ± 0.4, 10.0 ± 1.0, 23.0 ± 5.0 and 53.0 ± 7.0 $\mu g\,\ell^{-1}$. The standard deviation of the mean value encompasses the total variation of the seawater dosing system as determined by averaging all the biweekly measurements for cadmium.

## Results

The effects of cadmium on the two test species can be quantified by three interrelated parameters: survival, morphological aberrations, and reproduction.

Survival of both test species was affected similarly by exposure to cadmium. The 96-h LC50 for acute toxicity tests was 110 $\mu g\,\ell^{-1}$ for both species. In the life cycle exposure, survival data were analyzed (Table 1) by the Kruskal-Wallis K-sample test, in which animals were ranked (R = rank) by the number of days they survived (Lee 1980). Values in the table are the average rank of time to death for each treatment for each species. Because the test duration (51 days) and the number of animals at the start of the test (120) were the same for each species, the ranks can be directly compared. Underlining indicates those treatments which were not significantly different at $P = 0.05$. Therefore, survival in the lowest two concentrations and the control were not significantly different from each other; survival was significantly different between the higher two and the lower three concentrations. Ranking of each species pooled over all concentrations of toxicant showed no significant difference between species at $P = 0.05$.

*Table 1.* Percent survival of *Mysidopsis bahia* and *Mysidopsis bigelowi* in chronic exposures to cadmium (as $CdCl_2$) in a 51-day flow-through seawater test. Values (R) are average ranks per treatment per species determined by the number of days they survived. Twenty-four animals were originally placed in each treatment. Underlined values are not significantly different at $P = 0.05$ level.

| Cadmium exposure concentration, $\mu g\,\ell^{-1}$ | | | | | |
|---|---|---|---|---|---|
| Species | Control | 5.1 | 10.0 | 23.0 | 53.0 |
| R *M. bahia* | 84.3 | 94.8 | 69.8 | 39.3 | 14.3 |
| R *M. bigelowi* | 81.6 | 96.1 | 72.0 | 39.0 | 13.6 |

Survival curves for each species are plotted from the rank data (Fig. 1 and 2). At 23.0 and 53.0 $\mu g\,\ell^{-1}$ cadmium, survival decreased rapidly for both species to zero by day 27. At 0.0 and 5.1 $\mu g\,\ell^{-1}$ the curves are gradual and begin to level off around day 40. Percent survival in the 10.0 $\mu g\,\ell^{-1}$ concentration also declined gradually until day 30, when it decreased rapidly, indicating a quite different pattern for this level of exposure. In fact, survival at the 10.0 $\mu g\,\ell^{-1}$ concentration is significantly different from the control at $P = 0.11$.

Morphological aberrations were evident in both species exposed to cadmium at the onset of sexual maturity. External genitalia in males were aberrant in their position on the animals being widely separated or undeveloped (Fig. 3 and 4). Females failed to develop brood pouches (Fig. 5 and 6). Both sexes developed a carapace malformation in which the carapace pulled away from the metasome, creating a wing-like appearance that apparently interfered with molting (Fig. 5 and 6). Many females died

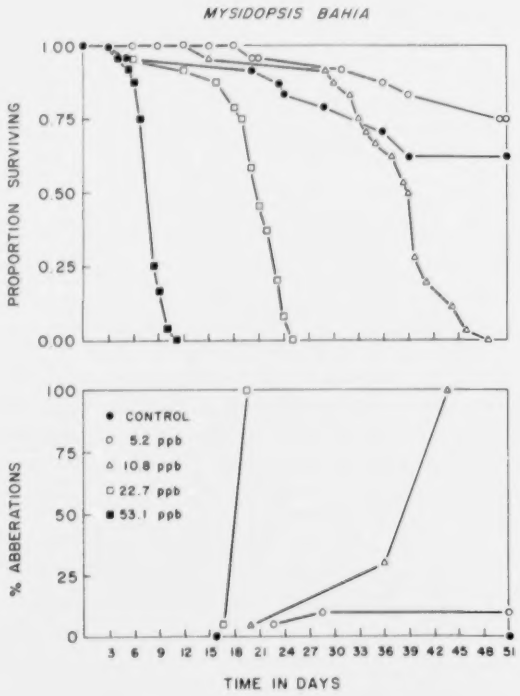


*Fig. 1.* Survival and percent occurrence of aberrations of *Mysidopsis bahia* exposed to cadmium (as $CdCl_2$) in a 51 day flow-through seawater test.

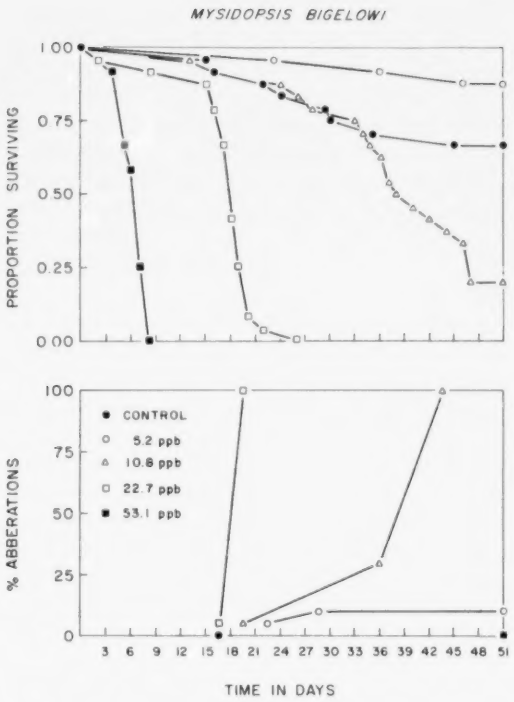


*Fig. 2.* Survival and percent occurrence of aberrations of *Mysidopsis bigelowi* exposed to cadmium (as $CdCl_2$) in a 51 day flow-through seawater test.

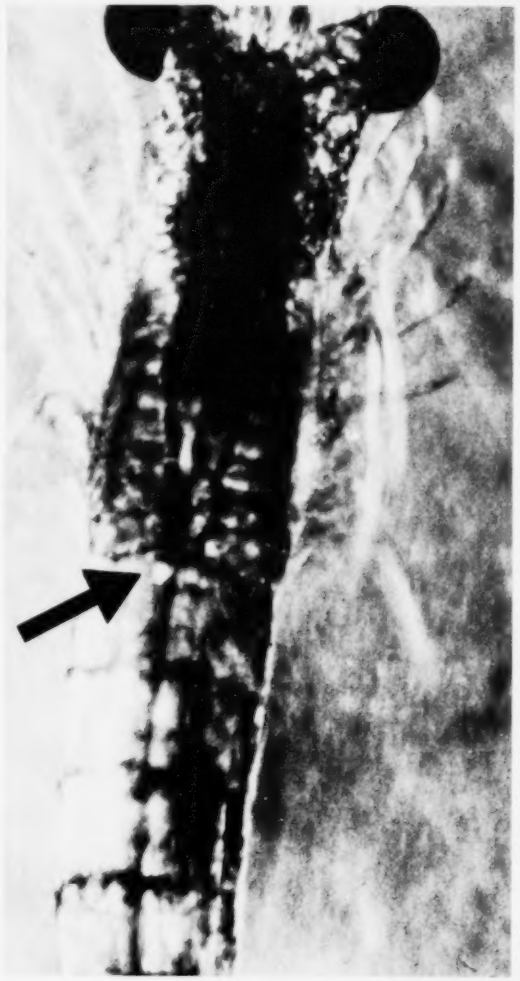

*Fig. 3.* Control male *M. bahia* on day 35 showing normal gonad development (arrow).

while molting after the release of the initial brood. Necrosis of tissue on the tips of the antennae and thoracic legs occurred on both sexes as well (Fig. 7).

As with survival, the incidence of aberrations was similar for both species. The onset of sexual aberrations and the percentage of the test populations afflicted at each toxicant concentration are illustrated in Figs. 1 and 2. The inverse relationship between onset and percentage of aberrations versus survival is apparent for *M. bahia* (Fig. 1). *M. bigelowi* has an identical relationship (Fig. 2).

Time of onset of morphological aberrations could be directly correlated to the cadmium concentration. At the high concentration (53.0 $\mu g\,\ell^{-1}$) all the animals died by day 11 and did not survive long enough to demonstrate aberrations. Deformities at 23.0 $\mu g\,\ell^{-1}$ appeared on day 16 and 100% of the animals were affected by day 20. At 10.0 $\mu g\,\ell^{-1}$, deformities appeared on day 19, and 30% of the animals were affected by day 36. By day 43, all the animals at this concentration had died. Only 5% of the animals at 5.1 $\mu g\,\ell^{-1}$ were deformed on day 22 and 10% were deformed by day 43. No malformations were noted in the controls at any time for either species.

The cumulative reproductive rate for each species, as measured by the number of young produced divided by the number of available reproductive days, is shown in Tables 2 and 3. Inhibition of reproduction at the 53.0 and 23.0 $\mu g\,\ell^{-1}$ is a direct reflection of the mortality of the test animals. In

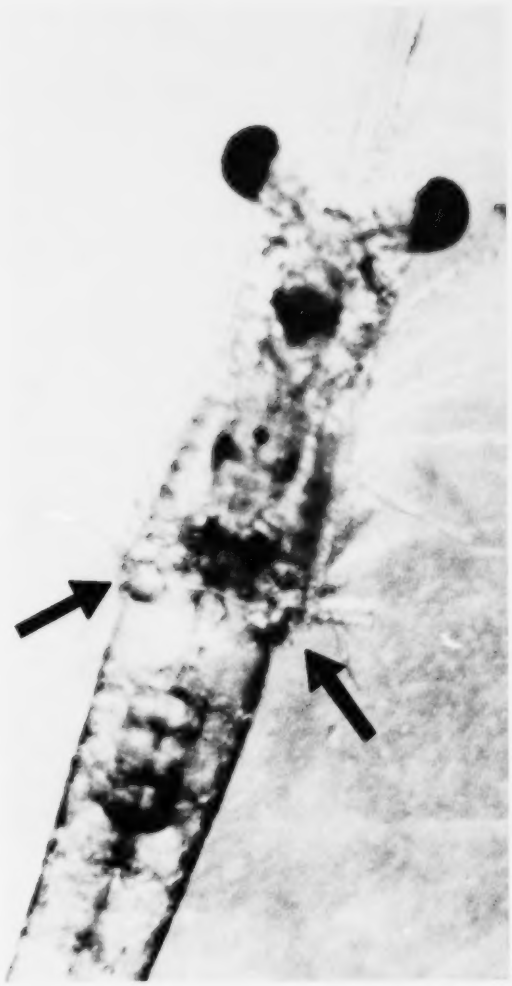

*Fig. 4. M. bahia* male exposed to 5.1 $\mu g\,\ell^{-1}$ cadmium (as $CdCl_2$) for 29 days showing aberrant gonad development (arrows).

*Fig. 5.* Control female *M. bigelowi* on day 35 with normal brood sac filled with developing embryos (arrow).

both species, all animals had died by day 11 at 53.0 $\mu g\,\ell^{-1}$ and by day 25 at 23.0 $\mu g\,\ell^{-1}$.

At 10.0 $\mu g\,\ell^{-1}$, the cumulative reproductive rate was initially enhanced in both species (Tables 2 and 3). It was not, however, significantly higher than the control at $P = 0.05$. As the percentage of abnormalities increased, further reproduction was inhibited at this concentration. There was no significant difference in cumulative reproductive rate between the control and the 5.1 $\mu g\,\ell^{-1}$ concentration for either species.

## Discussion

In this study, the two species demonstrated identical effects due to toxicant concentration, type of response and survival curves for acute and chronic assays. Based on these results, *M. bigelowi* is considered a suitable organism for marine toxicological studies, especially when dealing with pollution problems in temperate waters. Recent studies (unpublished data) indicated that temperature is more critical to *M. bigelowi's* productivity than to that of

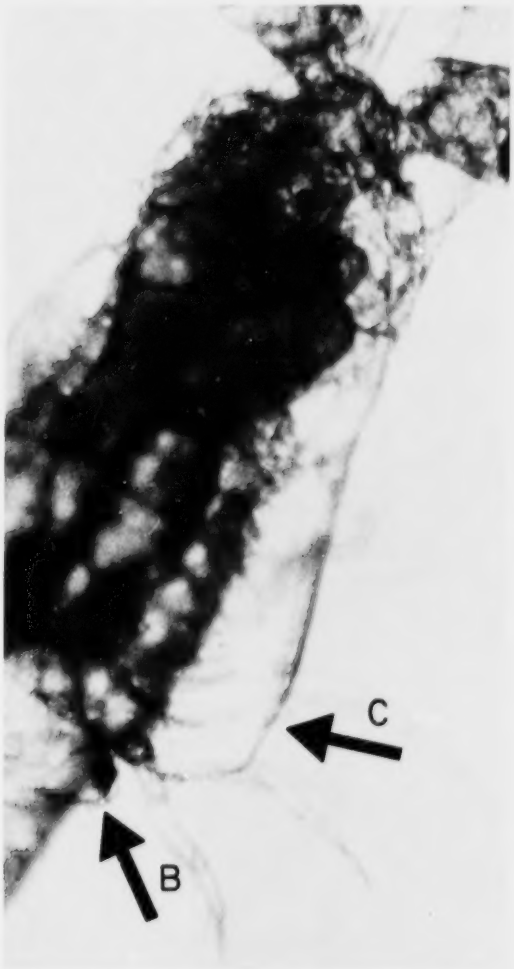


*Fig. 6. M. bigelowi* female exposed to 10.0 $\mu g\,l^{-1}$ cadmium (as $CdCl_2$) for 23 days with undeveloped brood sac (B) and malformed carapace (C).

*M. bahia*. There was a direct relationship between increased temperature (in the 19–25 °C range) and the number of young produced. The present cadmium assay, at 21 ± 1 °C, was below the optimum temperature range for productivity in *M. bigelowi*. This may account for the lower reproductive rate for *M. bigelowi* in this study.

Comparison of our results with those of Nimmo et al. (1977, 1978) show an effect of salinity on the toxicity of cadmium. With our test conditions (21 °C and 30 ‰ salinity), the 96-h LC50 was 110 $\mu g\,l^{-1}$ and the chronic effect concentration in the life cycle test was 10.0 $\mu g\,l^{-1}$ for *M. bahia* and *M. bigelowi*. Nimmo *et al.* calculated a 96-h LC50 of 15.5 $\mu g\,l^{-1}$ at 25–28 °C and 10–17 ‰ salinity. In the

*Table 2.* Reproduction of Mysidopsis bahia during chronic exposure to cadmium (as $CdCl_2$) in a 51-day flow-through seawater test. Available female reproductive days are calculated by dividing the total number of sexually mature females by the number of days they survived.

| Measured cadmium concentration, $\mu g\,l^{-1}$ | | | | | |
|---|---|---|---|---|---|
| Parameter observed | ≤0.3 Control | 5.1 | 10.0 | 23.0 | 53.0 |
| Percent survival | 62 | 83 | 12 | 0 | 0 |
| Days to 1st brood Release | 27 | 23 | 23 | – | – |
| Available female reproductive days | 206 | 302 | 186 | – | – |
| Total spawns | 17 | 16 | 27 | – | – |
| Total young | 80 | 80 | 122 | – | – |
| Total number young per available female reproductive day | 0.39 | 0.26 | 0.66 | – | – |
| Total number broods released per available female reproductive day | 0.88 | 0.05 | 0.15 | – | – |

*Table 3.* Reproduction of *Mysidopsis bigelowi* during chronic exposure to cadmium (as $CdCl_2$) in a 51-day flow-through seawater test. Available female reproductive days are calculated by dividing the total number of sexually mature females by the number of days they survived.

| Measured cadmium concentration, $\mu g\,l^{-1}$ | | | | | |
|---|---|---|---|---|---|
| Parameter observed | ≤0.3 Control | 5.1 | 10.0 | 23.0 | 53.0 |
| Percent survival | 62 | 92 | 42 | 0 | 0 |
| Days to 1st brood release | 33 | 32 | 27 | – | – |
| Available female reproductive days | 210 | 294 | 122 | – | – |
| Total spawns | 4 | 5 | 4 | – | – |
| Total young | 17 | 16 | 13 | – | – |
| Total number young per available female reproductive day | 0.08 | 0.05 | 0.11 | – | – |
| Total number broods released per available female reproductive day | 0.02 | 0.22 | 0.03 | – | – |

*Fig. 7.* Swimming legs of *M. bahia* male exposed to 23.0 $\mu g\ l^{-1}$ cadmium (as $CdCl_2$) for 23 days showing necrotic tissue within the exoskeleton (arrows).

23-day life cycle test, they reported decreased survival at 10.6 $\mu g\ l^{-1}$ with 20–28 °C and 15–23 ‰ salinity. Comparison of these studies indicates an increased lethality rate at low salinity and high temperature in the 96-h acute tests but not in the chronic sublethal tests.

O'Hara (1973) and Vernberg *et al.* (1977) found that high temperature and low salinity increase the uptake rate of cadmium. O'Hara showed that conditions of 30 ° and 10 ‰ salinity increased cadmium toxicity to the fiddler crab *Uca pugilator*. He found that although the effect of temperature was more pronounced than that of salinity, temperature had less effect at high salinities. O'Hara postulated

that these salinity effects were the result of increased ionic regulatory activity at low salinities due to accelerated uptake of ions. Vernberg *et al.* observed that as salinity decreased, mortality increased in the shrimp, *Palaemonetes pugio*.

Theede *et al.* (1979) and Jones (1975) found less tolerance to cadmium at low salinities and high temperatures and concluded that species living near their distributional limits are sensitive to lower pollution levels than those living at optimum environmental conditions. Theede (1980) later observed that sensitivity to chronic exposure to cadmium is less dependent on environmental salinity and temperature than are the results of acute tests which use high cadmium concentrations.

The effects on reproduction in this study, although occurring at the same toxicant concentration as reported by Nimmo *et al.* (1977, 1978), appeared to be due to an indirect mechanism rather than to direct toxic effects. Where we found exoskeletal malformations and retarded gonadal development, Nimmo *et al.* found delay of brood release and a sharp decrease in the number of young released per female. D'Agostino & Finney (1974) found that cadmium inhibited development of ovigerous females in the copepod *Tigriopus japonicus*.

Several studies have addressed the effects of cadmium on skeletal malformation in fish and invertebrates. Muramoto (1981) found that fish accumulated cadmium in the vertebrae and lost calcium and phophorus resulting in malformation due to demineralization. This interaction of 'competition' of cadmium with calcium is also suggested by Theede (1980) in the context of deterioration of the veliger shell structure under the influence of cadmium. It appears from our present study that cadmium may affect the molting process by competing or interfering with the calcification of the new molt. The blood calcium concentration of an invertebrate is insufficient to provide the necessary calcium for the new molt; therefore, calcium must be taken up from the external medium (Lockwood 1967). If a high concentration of cadmium ions is available in the medium, it may interfere with the calcification process, resulting in a soft cuticle that is subject to distortion. Jennings & Rainbow (1979) found that the crab, *Carcinus maenas*, accumulated cadmium in proportion to the concentration and length of exposure. The exoskeleton contained 59–80% of the absorbed cadmium. Vernberg *et al.* (1977) noted that cadmium uptake increased as salinity decreased in *Palaemonetes pugio*. Similar effects of salinity on uptake were shown in the studies of Wright (1977) and Hutcheson (1974). Wright found that in the crab, *Carcinus maenas*, cadmium was heavily accumulated by the carapace and uptake in 50% seawater was much faster than in 100% seawater. Hutcheson observed a fourfold increase in accumulated cadmium in the carapace of blue crabs, *Callinectes sapidus*, in animals held in 17% seawater as compared with those in 100% seawater. However, no malformations in the exoskeleton of *M. bahia* at low salinity exposure were reported by Nimmo *et al.* (1977, 1978). Thus the question remains whether other factors may be involved in the production of this anomaly.

In addition to the possible effect of cadmium on calcification during molting, there is also some evidence of the interference by cadmium in the osmotic processes involved. Many of the mortalities noted in this study were animals found dead in the process of molting. Cadmium has been shown to inhibit enzymatic activity or otherwise interfere with physiological processes (Hochachka & Somero 1973). Water uptake via osmosis stretches the new cuticle and splits the old molt. In the shrimp, *Palaemonetes pugio*, high body burdens of cadmium inhibited molting (Vernberg *et al.* 1977). Osmosis depends on a premolt rise in osmotic pressure in the blood due to increases in the concentration of calcium and magnesium. Some of the necessary calcium comes from the blood, the remainder from the medium. While there are no studies with invertebrates, Larsson *et al.* (1981) reported that potassium and calcium concentrations in blood plasma of flounder demonstrated a dose-dependent depression after exposure to sublethal concentrations of cadmium.

To maintain the normal osmotic pressure within the cells during molting, an increase in the soluble organic components of the cells is accompanied by water uptake (Lockwood 1967). Hochachka & Somero (1973) suggest that most of the osmotic adjustments of tissue cells are due to variations in the concentrations of intracellular amino acids. In crustaceans, the major contribution to changes in total intracellular osmolality is made by glycine, alanine, proline and glutamate. The enzyme glutamate dehydrogenase is now believed to be closely

involved in cell volume regulation (Hochachka & Somero 1973). Glutamate dehydrogenase is a zinc containing metallo-enzyme, the metal being directly involved in the reaction. Pool (1981) refers to *in vitro* experiments that have shown the replacement of zinc by cadmium. Copius Peereboom (1981) discussed the possibility of the replacement of zinc by cadmium creating a zinc deficiency and resultant inhibition of enzyme activity. An effect of this sort on enzymatic processes could be responsible for disruption of the molting process.

In summary, acute and chronic responses of the two congeneric mysid species to cadmium toxicity were essentially identical. This study also demonstrates the interaction of both acute and chronic toxicological responses with the environmental variables salinity and temperature. The mechanism of cadmium toxicity is a complex phenomenon potentially involving physiological processes of skeletal calcification and osmoregulation.

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